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# REPLY

We would like to thank Professor Lehmann for his valuable comments in which he raises 3 points. First, we agree that left ventricular hypertrophy (LVH) and its increased risk of heart failure, as well as coronary atherosclerosis, are important risk factors for sudden cardiac death (SCD). Therefore, we adjusted our analyses for incident heart failure and for several measures of atherosclerosis. These variables were not available in the earlier analyses with baseline information from the Rotterdam Study, but in the current analyses we were able to adjust for this follow-up information.

Second, in earlier studies we reported on the prognostic value of an abnormal T-wave axis (1). In these analyses, QTc prolongation was not an independent risk factor, but this was probably due to a lack of sample size and to the fact that we now used SCD as an end point instead of sudden death and all cardiac death. Moreover, we used other cut-off points for QTc prolongation than in our earlier study. Because of the substantial interest of regulatory bodies for the potential role of QTc prolongation and because of the widespread clinical use of the QTc interval (whereas the frontal T axis is uncommonly used in clinical practice), we focused our current analyses on this ECG feature.

In a model containing the QTc interval, T-wave axis, LVH on the electrocardiogram, age, and gender, an abnormal frontal T-wave axis (between −15° and −180° or between 105° and 180°) showed a strongly increased risk of SCD (hazard ratio [HR] 2.84 [95% confidence interval {CI} 1.37 to 5.90]), but the HR of QTc prolongation was still 3.16 (95% CI 1.69 to 5.89). In the fully adjusted model, this HR hardly changed. Additional adjustment for QRS duration (≥120 ms) resulted in an HR of QTc prolongation for SCD of 2.97 (95% CI 1.51 to 5.83).

Third, antiarrhythmic drugs may increase the QTc interval, but as they then precede the association between QTc interval and SCD they should not be adjusted for. We conclude that QTc prolongation in our study was an independent risk factor for SCD (2).

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